among Colorado cows that it is almost a curiosity; and when it does occur, it is usually either in animals that have had the worst of care as regards overcrowding or in imported animals; native cattle are nearly immune. The testimony from all over our high dry plateau, at from five to eight thousand feet altitude, is the same. So, if it is true that consumption is very often contracted by drinking milk from consumptive or tubercular cows, the fact of Colorado's cattle being so remarkably exempt from this disease only adds another factor to our safety. If only two cows to the hundred have consumption in Colorado and twenty-five to fifty to the hundred have consumption in the East and elsewhere, there is not much room for doubt as to the comparative danger from milk infection. In a drink of milk taken in any of our average cities below two thousand feet altitude we run about 30 per cent. more risk of taking consumption than we do from a glass of milk in Colorado."

An interesting question is, Will such an immunity as exists, for example, at Colorado Springs remain constant? I think that it will, for the following reasons: We know that, as regards indoor occupation, contact with tubercular invalids, and lack of sanitary precautions, the maximum danger has been about reached. For some years past all these factors have had full opportunity to exert their baneful influence, and the results show for 1896 no increase in non-imported consumption; and in the future, although it is true the city may become more crowded, this fact or danger will be clearly met and defeated by the sanitary precautions enforced, as a law passed in 1897 fines any one from one to five dollars for expectorating on the sidewalks; and the sanitary condition of the city will be constantly improved to such a degree that it is safe to predict that we can not only keep our present low percentage of infection from tuberculosis, but will even lower it in the future.

MULTIPLE SCLEROSIS IN CHILDHOOD,

WITH A REPORT OF THREE CASES.

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The first case of disseminated sclerosis occurring in a child was published by Schuele in 1871. It is at the same time one of the three cases of the kind in which an autopsy confirmed the clinical diagnosis. Schuele's case was followed by the publication of a number of English observations (Dreschfeld, Pollard, Bristowe, etc.), and in 1879 by the report of two cases from Erb's clinic by Ten Cate Hoedemaker, who, in reviewing the cases previously recorded, only accepted Schuele's and

Dreschfeld's cases as satisfactory. In 1883 Marie[18] wrote a paper on the subject, collecting in all thirteen cases from literature and adding a case observed by Charcot and himself; his list at the time included the cases Hoedemaker had refused to accept. Unger,[40] in Vienna, published in 1887 an elaborate monograph upon the occurrence of multiple sclerosis in children, and increased Marie's list of cases to nineteen, one of them a case of his own observation. A number of publications reporting one or two cases of the kind followed in the German, French, and English medical journals. In 1893 Totzke, a pupil of Mendel, in Berlin, published two new cases in a dissertation, and reviewed all the cases published after the appearance of Unger's monograph, tabulating thirty-nine cases up to date. In this country Pritchard[35] wrote a chapter on multiple sclerosis in Keating's *Encyclopædia of the Diseases of Children*, and Sachs[37] devotes a chapter to the disease in his book on *The Nervous Diseases of Children*, bringing the subject up to current times.

Having had the good fortune to observe at the Mount Sinai Hospital Dispensary and in my private practice three cases of the disease in children during the past year, I was led to inquire into the frequency of its occurrence at this age and to study the special literature on the subject. This study has forced the conviction upon my mind that the diagnosis of multiple sclerosis in a number of the cases published as such could be fairly challenged, and that the various statistical tables include cases which should properly be classified under other headings. It is not the purpose of this paper, however, to pass judgment, case for case, upon the previously reported cases; that would hardly repay the effort. Having missed in the previous writings—even in Unger's valuable monograph—an exhaustive discussion of the diagnosis of the disease as it occurs in children, I propose, after submitting the histories of my three cases to your kind criticism, to discuss at some length this very point—the differential diagnosis. Inasmuch as recent years have added much to our knowledge of the nervous diseases of children, new types having been created and old ones modified, an effort in this direction, I trust, will not be devoid of interest.

CASE I.—Anna F.,* of German parentage, nine years of age, the eldest of five children; was born spontaneously after somewhat prolonged labor. The child is said to have been blue in the face at its birth, but began to cry after a few moments. Its mother is a large, robust, perfectly healthy woman, and has never miscarried. The father is of good health, and has been a moderate drinker of beer. Of the five children, one died of scarlet fever and one of croup. There is no history of syphilis, nor does the examination of the mother and her three children reveal any indication of the disease. The other two children living are in perfect health, and show no signs of the disease the patient is suffering from.

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* The patient was presented on October 23, 1896, before the Society of German Physicians, and was seen there amongst others by Drs. Sachs, Jacoby, and Leszynsky.

Previous History.—When the child was three months old a number of furuncles broke out on the face and neck, beyond which they did not spread. Starting from one of these an attack of erysipelas developed, which spread over the face and scalp. It had hardly subsided when a relapse occurred. In all the child was ill for three weeks, high fever accompanying both attacks. At no time were there any convulsions. The mother states that soon after she noticed that the child's hands trembled. The patient was two years old before she learned how to walk and talk, the younger children learning both when nine to twelve months old. The gait of the child did not differ from that of other children. Her speech has always been slow. Two years ago she was taken with a severe attack of scarlet fever, and was sick for six weeks. For three months thereafter her speech was very much worse than before; it was so bad that her mother had great difficulty in understanding her. Then her speech gradually improved, and at present it is not much worse than before. The tremor in the hands increased after the scarlet fever. The child goes to school, but has great difficulty in "keeping up." She does not learn nearly as well as her younger brother and sister. "She is of good disposition, but not bright," according to her mother's statement. She laughs and weeps very readily, and at times immoderately, without any apparent motive.

On October 17, 1896, she was brought to me at the Mount Sinai Hospital Dispensary.

Physical Examination.—The child is rather small for her age, but otherwise well developed and in good general health. The pupils are equal, and respond well to light and accommodation. There is no nystagmus and no ocular-nerve palsy. The fundus and vision, kindly examined by Dr. Koller, are perfectly normal. There is considerable hypermetropia.

There is a slight tremor of the tongue, which is protruded straight. Facial innervation is equal on both sides. Taste and hearing are good. There is a slight general weakness of the muscular frame; nowhere any rigidity. The deep reflexes of the arms are lively, the *knee-jerks* are both *exaggerated, ankle-clonus* can be occasionally obtained from the right foot; the Achilles-tendon reflex of the left foot is very active. There is a very marked *intention tremor* present in both hands, more in the right than the left. In seizing a glass of water and attempting to drink from it the tremor is very characteristic. When the glass is full the patient is apt to wet her clothes. The amplitude of the tremor increases somewhat as the destination is approached. The degree of tremor varies considerably according to the child's emotional condition. It is very noticeable when she attempts to write. Several characteristic specimens of her handwriting were obtained.

The patient's gait is easy; there is neither ataxy nor spasticity noticeable. In running she is not as quick as other children of her age. There is no muscular atrophy, and the electric examination shows normal conditions. Her voice is *hoarse,* and has always been so; it breaks easily. A laryngoscopical examination has not been possible as yet. Her *speech* is *slow* and *monotonous* in intonation. Mentally the child is backward and deficient. In conversation she impresses one as a child of four or five would, and not even as a bright child of that age. Her internal organs are sound; the urine is free from sugar and albumin; the bladder and rectal functions are normal.

To recapitulate briefly the conditions presented by this case, we have a child of nine years who suffered a severe attack of erysipelas when three months old and an equally severe attack of scarlet fever when seven years old. At the present day we find a marked *intention tremor* of the hands, *exaggerated knee-jerks,* and an occasional *ankle-clonus.* The *voice* is *hoarse, speech slow* and *monotonous.* The child is of impaired mental capacity, cries and laughs easily.

For the present suffice it to state that this symptom complex is characteristic of multiple sclerosis. A few points concerning the differential diagnosis will be referred to later on.

CASE II.—Bertha S., eleven years old, of American parentage, was brought to my office in September, 1895, and was seen but a single time, as she lives in another town of this State. She is the second of two children. The older child is perfectly well. She was born at term and without difficulty. Her mother is of a nervous temperament, of average health. On the father's side there has been no indulgence in liquor and no syphilis. There is no history of previous nervous or mental disease in the family. The child was always regarded as nervous, but never had convulsions. Her eyes have always been prominent. Some years ago she suffered for some weeks from complete *ptosis of the left eyelid,* which disappeared under electric treatment. She never complains of headache, and does not vomit. There is occasional *vertigo.* For the past year she has lost control of her bladder to a great extent; at times she wets herself without knowing what is going to happen; at other times she is unable to retain her water when she feels it wants to come. At the very best she must hasten to empty her bladder in order not to wet herself. She has been *weak* and *unsteady on her legs* for the past two or three years. Of late her gait has been getting worse.

Physical Examination.—The girl is fairly well developed, of good size for her age. The head is large, rhachitic in shape, 54 cm. in circumference. The eyes are prominent, as in exophthalmic goitre. There is divergent squint, owing to weakness of the right internal rectus. There is a fine *nystagmus* while the eyes are at rest; upon extreme excursions the nystagmus increases and becomes *jerky* in character. The ophthalmoscopic picture shows optic atrophy in both eyes, more advanced in the right eye. Vision: O. D.=10/XLV; O. S.=10/XLV. Not improved by glasses. The visual field was not determined. The pupils are large and respond to light and accommodation. There is a fine tremor of the fingers while the patient is at rest. Upon motion there is a very marked *intention tremor* (finger-pointing, glass of water tests). The tremor is about equal in both hands. There is no loss of power and no rigidity in the arms. The gait is that of an *ataxic paraplegia,* with a very evident *cerebellar grogginess* added. The child sways when standing with her eyes open, and is unable to stand with them closed. The *knee-jerks* are greatly *exaggerated.* There is right *ankle-clonus,* on the left side an increased reflex from the Achilles-tendon. There is some rigidity in the legs, but no contractures. The various special tests show considerable ataxy and disturbance of the muscular sense in the legs. Sensation is perfect in all parts of the body. The examination with the faradic current shows slightly increased excitability in the muscles of the legs, otherwise normal conditions. The child is of good intelligence; is easily frightened, trembles when nervous. Speech is not affected. The pulse is 84, and the internal viscera are sound. There is no tenderness to percussion in any part of the head.

The morbid conditions presented in this case are: *optic atrophy, strabismus, nystagmus, and previous ptosis* as eye symptoms. *Intention tremor,* some *rigidity* of the legs, *increased knee-jerks and ankle-clonus, a cerebellar ataxo-paraplegic gait,* the Romberg symptoms, and loss of control of the sphincter of the bladder. The observation is in some respects, I am well aware, imperfect, as is almost always the case when but a single examination has been possible. Nevertheless, the clinical picture is sufficiently clear to allow a positive diagnosis, the diagnosis having to take account of multiple sclerosis, Friedreich's ataxy, ataxic paraplegia, tumor of the brain, and hereditary cerebellar ataxy. Friedreich's ataxy can be excluded by the exaggerated knee-jerks, and ataxic paraplegia by the eye-symptoms. The reasons for excluding brain tumor and hereditary cerebellar ataxy will be given later in the course of the discussion of the diagnosis in general.

CASE III.—Anna F., aged fifteen years, of Russian parentage, was first seen at the Mount Sinai Hospital Dispensary, March 30, 1896, and was observed for some time. The family history is negative. She was born at term without any difficulty. She was always well until she was seven years of age, when she was taken ill with a severe cold, which, from the description given, may have been an attack of influenza. Shortly after this she began to have difficulty in walking, becoming unsteady on her feet, and tiring more quickly than before. At the same time her *speech,* which had been fluent, began to be *slow* and *difficult,* and her *fingers* began to *tremble* so that she could no longer sew, had difficulty in buttoning her clothes, and could not eat soup from a spoon without spilling. Her *legs* gradually became *stiff,* and the tremor in her hands has increased during the past year. She is subject to *vertigo,* which is, however, never very severe. No headache. There never has been any ocular nerve palsy and no vesical trouble.

Physical Examination.—The girl is mature for her age, broad-shouldered, and in robust general health. Her voice is harsh, and in her *speech* there is a *noticeable effort* as she slowly utters syllable for syllable with little or no variation of intonation. Her voice is also apt to break. There is no nystagmus; the pupils are equal and respond well. The ophthalmoscopic examination shows normal disks with some congestion of the veins (Dr. Koller). There is a classical, *oscillatory tremor* in her hands, which appears as soon as she moves them for any purpose. In attempting to drink from a glass of water she spills the water if the glass is more than half filled. The tremor is more vehement when the patient is nervous; at such times her head shakes. The deep reflexes of the arms are very lively. The gait is that of a *spastic paraplegia;* her toes do not, however, cling to the floor; at times there is some swaying. No Romberg. The *knee jerks* are greatly *exaggerated;* there is *ankle-clonus* on both sides, and there is some rigidity in the legs. The patient is of average mental capacity.

The *laborious speech,* the *intention tremor,* and *spastic paraplegia* in this case were so classically developed that in spite of the absence of eye-symptoms the diagnosis of multiple sclerosis could be made without hesitation.

DIAGNOSIS.—Naturally the differential diagnosis of multiple sclerosis in childhood must follow somewhat different lines than those laid down for the same disease in the adult. From a study of the cases recorded, and of my own cases, I find that the following diseases may have to be considered in the presence of a given case:

1. Infantile cerebral palsy.
2. Syphilis of the cerebro-spinal axis.
3. Hereditary spinal ataxy (Friedreich).
4. Hereditary cerebellar ataxy (Marie).
5. Acute disseminated myelitis (Leyden).
6. Tumor of the brain.
7. Hysteria.

1. Increased myotatic irritability, spastic paralysis, and rigidity, representing various degrees of one and the same morbid condition, confined to the legs or involving all four limbs; mental impairment; partial or complete hemiplegia and strabismus are conditions common to both multiple sclerosis and the infantile cerebral palsies. In his monograph on *Cerebral Diplegias*, Freud furthermore points out that the symptoms considered most characteristic of multiple sclerosis—the intention tremor, the nystagmus, and the bradylalia—are occasionally observed in cases bearing all the marks of a cerebral diplegia. It is true it is comparatively rare that we find more than one of these three symptoms present in a case of this kind, and there is no instance on record in which all three were observed. Nevertheless, if they occur singly or two at a time, Freud argues, there is no reason why they should not occasionally be seen simultaneously; nor are the cases of multiple sclerosis common in which this symptom-triad is complete. Of the thirty-five cases reported in children I have found but nine cases (Schuele, Charcot-Marie, Hoedemaker, Dreschfeld, Totzke, Schoenfeld, and Unger) in which intention tremor, nystagmus, and bradylalia were noted. The many points in common between the two morbid conditions indicate the difficulties that may be encountered in attempting the differential diagnosis; and these difficulties are enhanced by a common etiology, both occurring after the acute infectious diseases. I have emphasized the close resemblance possible in the clinical picture of these two diseases for the reason that in a number of the cases recorded as multiple sclerosis in children the diagnosis of diplegia would have been more justifiable. Marie has voiced this view already by confessing in his *Leçons sur les Maladies de la Moelle*, in 1892, that his series of fourteen cases, published in 1883, included quite a number (*un bon nombre*) which he would to-day classify under the heading of infantile cerebral palsies. In looking over all the cases reported I find that those of Dick-

inson, Sparks, Humphreys, Cheadle, Bodson, Railton, probably one of Freund's and one of Bruns's cases, and perhaps two or three others, are cases of this kind. The majority of these were published at a time when a knowledge of the great frequency and of the clinical features of infantile cerebral palsies had not yet become common property through the writings of Gowers, Marie, Freud, Osler, Sachs, Peterson, and others..

To avoid any misunderstanding, it will be well to state here that in the large majority of cases of infantile cerebral palsy the question as to the presence of disseminated sclerosis will not arise at all; the boot is rather on the other leg—that is to say, in every case of supposed multiple sclerosis in childhood, and especially in *early* childhood, the far more common condition of infantile cerebral palsy will have to be excluded.

In spite of the close resemblance in the symptom complex of the two diseases which has been pointed out, in most if not all cases a differential diagnosis will be well possible. In this respect a consideration of the mode of onset and of the course of the disease is of supreme importance. Infantile cerebral palsy is either congenital or a disease of acute onset; in the congenital or birth-palsies a history of difficult labor, instrumental delivery, or premature birth is of great diagnostic weight. Such a history should make us exceedingly chary of making the diagnosis of multiple sclerosis in early childhood. In these cases there may be an apparent development of the symptoms as the child grows older and the disorders of motion and speech and the mental impairment become manifest. In the acquired forms of the disease, in which acute infectious diseases, endocarditis, and convulsions are the main etiological factors, the symptoms produced by the acute lesion make their appearance immediately, and there is little or no further development; on the contrary, there may be some retrogression in the symptoms, very much as in the cerebral palsies of adult life. Although the morbid process in infantile cerebral palsies may not be absolutely stationary, but may, according to Marie's views, continue as an eccentric secondary sclerosis, it is always localized—*i. e.*, it may involve immediately adjacent areas, but cannot affect distant parts of the cerebro-spinal axis.*

In disseminated sclerosis the conditions are quite the reverse. Occasionally the onset may be acute or subacute, especially in the cases which develop immediately after an acute infectious disease; but the morbid process, once started, does not cease with the first onset. It breaks out afresh again and again in any part of the cerebro-spinal axis and its nerves, adding new symptoms to the old. Another point of distinction of considerable value is the fact that many of the symptoms in multiple sclerosis show a very marked tendency to improve and disappear. This is especially true of the ocular nerve palsies, of which we have a good illustration in the temporary ptosis noted in Case II. As Oppenheim has pointed out, the tendon reflexes, the field of vision, and the disturbances of sensation may vary even from examination to examination. To express briefly what has been explained at length, *multiple sclerosis* is a *progressive* and *retrogressive*, *infantile cerebral palsy* a *stationary* disease.

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* New foci of disease may, however, develop in the presence of an endocarditis, causing recurrent embolism, or by fresh hemorrhages occurring during convulsions. Such an occurrence is very rare, and may have given rise to the diagnosis of multiple sclerosis in the case of Humphreys.

When the mode of onset and the course of the disease do not throw sufficient light upon a case, other points may furnish the desired key. In Case I. the history of a somewhat prolonged birth (first child) would justify the suspicion that it is a case of infantile cerebral palsy. Beside the fact that the course of the disease, especially the increase of the symptoms after the scarlet fever, favors the diagnosis of multiple sclerosis, I believe the following point is conclusive: In cerebral diplegia the legs are never less affected than the arms. As a general rule, they are more affected. In our case the arms are much more involved that the legs.*

Optic atrophy,† especially partial discoloration of the disks, which is almost pathognomonic of multiple sclerosis, central scotoma, or contraction of the field of vision, may furnish decisive evidence in a doubtful case.

It will be seen that no reference has been made to the presence of the cardinal symptoms of disseminated sclerosis as bearing any weight in the differential diagnosis. In this connection it may be stated that intention tremor, nystagmus, and bradylalia are the rule in multiple sclerosis and the exception in infantile cerebral palsies. In the presence of two or three of these symptoms, providing they are present beyond cavil, it will require strong evidence to the contrary to throw out the diagnosis of multiple sclerosis.

2. It is well known that in the adult *multiple cerebro-spinal syphilis* may produce conditions which it is exceedingly difficult to distinguish from multiple sclerosis, so much so that so able an observer as *Oppenheim*, who has done much to further our knowledge of the clinical course of both these diseases, only recently published some cases through *Cassirer*,[43] in which a positive differential diagnosis was not possible. Although rare, cases of this kind may occur in children, as is shown by three cases published by *Moncorvo* in an article entitled "On the Etiology of Insular Sclerosis in Children, with Special Reference to the

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* Freud cites a somewhat similar case in a twin in his monograph, in which he makes the diagnosis of diplegia, p. 153. In his case there was rigidity of the legs.

† Optic atrophy is observed in rare cases of infantile cerebral palsy.

Pathogenic Influence of Hereditary Syphilis," which appeared in the *Revue Mensuelle des Maladies de l'Enfance*, 1887, vol. v. All of these three cases, occurring in children three or four years of age, and showing cutaneous lesions of hereditary syphilis, presented typical symptoms of insular sclerosis, and were diagnosed as such (Moncorvo). They all *recovered* under specific treatment. Although Moncorvo attempts to show the importance of syphilis as an etiological factor in the causation of multiple sclerosis, on the basis of these cases, we need not hesitate, I believe, in refusing to regard his cases as examples of the disease at all. They illustrate rather how well specific diseases may imitate the clinical picture of disseminated sclerosis even in the young. In one of the two cases of multiple sclerosis in children reported by Dickinson hereditary syphilis was present. There is no mention of specific treatment having been instituted.

It is not within the scope of this paper to discuss at length the differential diagnosis between multiple cerebro-spinal syphilis and disseminated sclerosis. That is a chapter in itself. Suffice it to say that the same considerations which enter into play in adults hold good in the young. In the latter we are apt to find, as in Moncorvo's cases, valuable evidence in the presence of the ordinary signs of hereditary syphilis.

3. *Hereditary spinal ataxy* shares with disseminated sclerosis a number of important symptoms—the nystagmus, the disturbance of speech, and an ataxic tremor of the extremities, which bears a certain though remote resemblance to an intention tremor. In the various articles on disseminated sclerosis in children the differential diagnosis between this disease and Friedreich's ataxy is entered into very fully. I believe that it is rarely difficult to differentiate between the two in practice. The main differences may be summed up as follows:

Friedreich's Disease.	*Multiple Sclerosis.*
Knee-jerks lost or diminished.	Exaggerated.
Eyes, pupils and fundus normal; ocular nerve palsies very rare.	Pupils frequently unequal, reflexes may be lost; discoloration of the optic disks; amblyopia; ocular nerve palsies common.
Speech halting and explosive, blurting.	Laborious, monotonous, and scanning.

Proof of heredity would be of little diagnostic value, as multiple sclerosis has been observed in more than one member of a family (Dreschfeld, Totzke, Leusch-Huber). At the Mount Sinai Hospital Dispensary I have seen multiple sclerosis in a man of thirty years, who stated that his father had suffered for many years from the same disease and had ultimately died of it.

4. According to *Marie*,[20] who first classified the atypical cases of hereditary ataxy published by Fraser,[8] Sanger Brown,[5] Nonne,[26] Klippel and Durante, and a number of other writers, giving them the name of *hereditary cerebellar ataxy*, to distinguish them from Friedreich's

disease, a distinctive feature of this type is its onset after the age of puberty. That it may occur earlier is shown by Fraser's original cases, in which it began in the third or fourth year. In Sanger Brown's twenty-one cases the disease began once each at the age of six, eight, eleven, thirteen, and twice at fourteen years—in all, six times before the age of fifteen. Recently Collins has published an interesting though atypical case in a boy of eleven years. The question as to whether multiple sclerosis or hereditary cerebellar ataxy is present may therefore arise in children as well as in adults, and does arise, for instance, in our second case.

The close similarity in the symptoms of these two diseases is evident in the following comparisons:

Hereditary Cerebellar Ataxy.	*Multiple Sclerosis.*
1. Gait, ataxic, groggy; feet wide apart.	1. (*a*) Spastic paraplegic; feet close together. (*b*) Ataxic, groggy; feet wide apart. (*c*) Ataxic paraplegic (*a*+*b*).
2. Station, Romberg's symptom absent.	2. Romberg may be present.
3. Arms, ataxy and some intention tremor.	3. Intention tremor; sometimes ataxy.
4. Oscillations and jerky movements of the head and trunk.	4. Oscillations and jerky movements of the head and trunk.
5. Exaggerated contractions of facial muscles during speaking.	5 Twitching in facial muscles during speaking.
6. Speech, hesitating and abrupt, or simply monotonous.	6. Laborious, scanning, or monotonous.
7. Eyes, jerky nystagmus; optic atrophy, contracted field of vision. The external recti muscles may be paretic or paralyzed.	7. Jerky nystagmus; optic atrophy, contracted field of vision; ocular nerve palsies.
8. Myotatic irritability increased; knee-jerks exaggerated, ankle-clonus; contractures and muscular rigidity.	8. Myotatic irritability increased: knee-jerks exaggerated, ankle-clonus; contractures and muscular rigidity.
9. Mental impairment in varying degrees.	9. Mental impairment in varying degrees.
10. Vertigo sometimes.	10. Vertigo common.
11. Vesical functions rarely affected.	11. Vesical functions more frequently disturbed.
12. Apoplectiform seizures do not occur.	12. Apoplectiform seizures occur in a small proportion of cases.
13. Heredity common.	13. Heredity uncommon.

On account of the close similarity in the symptoms of the two conditions, the differential diagnosis in a given case may be very difficult or even impossible. In Case II. the gait corresponded very well to that of hereditary cerebellar ataxy. It was ataxic, there was a tendency to reel, and there was some rigidity of the legs present. The increased knee-jerks, the optic atrophy, the amblyopia, and the jerky nystagmus all fitted well into this clinical type. The features which allowed the diagnosis to be made in favor of multiple sclerosis were (1) the history of a previous ptosis which disappeared under treatment; a temporary ocular nerve palsy is very characteristic of disseminated sclerosis. (2) The paresis of the internal rectus muscle. (3) The presence of the Romberg symptom, which is said not to occur in hereditary cerebellar ataxy.

(4) As supporting factors: the weakness of the sphincter of the bladder and the age of onset.

The absence of heredity is of no diagnostic value, as a number of isolated cases of hereditary cerebellar ataxy have been published. Some of them, it is true, may turn out to be cases of multiple sclerosis upon autopsy.

The difficulties the diagnosis may encounter are well illustrated in the following case, in an adult, which has been under my observation for more than two years:

Mrs. S., aged thirty years, born in Germany, was first seen at the Mount Sinai Hospital Dispensary on August 17, 1894. She has been married eight years and has had two children, both in good health and free from any nervous disorder. She has had four miscarriages, which were brought on intentionally. Her father died at the age of fifty-four years from hasty consumption. Her mother is fifty-nine years old, and in good health. A younger sister of the patient, though not very bright, and a brother are perfectly well. The patient's maternal grandfather was insane and committed suicide.

As a young girl the patient suffered from her fourteenth to her nineteenth year from chlorosis. Shortly after she had some weakness in her legs, especially in the right one, which, she says, she dragged; she had no pains in the legs. After two or three months, under turpentine rubbings, the trouble disappeared. Ever since, however, she states she has been a little unsteady on her legs. When she was twenty-seven years old the unsteadiness began to increase, and grew worse and worse. When she turns her head, while walking, she becomes so dizzy she is afraid of falling. She has never suffered the least pain in her legs or any other part of the body. No headache, occasional vertigo. During the past two years her hands have become unsteady, so that she cannot sew as well as formerly. She is clumsy in buttoning her clothes, and can write but with great difficulty. Both her legs and arms have grown weaker during this time. She cannot walk far without tiring. There has never been any bladder trouble.

Examination.—The patient is a well-developed woman with little adipose tissue. She walks with her feet wide apart and sways from side to side. Romberg is indicated. In the prone position there is marked ataxy of the legs, which is increased when the eyes are closed. At rest there is no tremor of the hands. Upon motion there is an irregular ataxic tremor, which is most apparent when the patient attempts to button her clothes. She fumbles about the button before she is able to catch hold of it. The tremor has not the oscillatory character seen in multiple sclerosis, and is of very much smaller amplitude. It occurs, however, only as an intention tremor. It is increased when the eyes are closed. The deep reflexes of the arms are very lively. The knee-jerks are exaggerated, and there is ankle clonus on both sides. The voice is clear; speech is deliberate, with monotonous intonation. In speaking there is excessive action of the labial muscles. The tongue is protruded straight, without twitching or tremor. The pupils respond well, and are equal in size. Upon looking upward there is rotary nystagmus in both eyes, more marked in the left. Dr. Koller kindly examined the patient's eyes and reported as follows: Vision: O. S. = 2/LX (fingers), emmetropic; O. D. = 5/XVIII, emmetropic.

Ophthalmoscopic Examination.—Left eye: The temporal half of the optic disk is excavated and of a bluish-green discoloration. The nasal half is more grayish. The disk seems diminished in size; the outlines are not very sharp. There is some increase of interstitial tissue along the larger bloodvessels. The calibre of the retinal vessels is unchanged. The retina is thin.

Right eye: The disk is of normal size, well outlined. The temporal half bluish-green and excavated; nasal half gray. The same changes are seen about the bloodvessels as in the left eye, only in a lesser degree." Dr. Koller adds that "this picture indicates a mixture of the two main types, primary atrophy and atrophy following inflammation. It may be looked upon as a primary atrophy with increased interstitial tissue growth." The visual fields show irregular contraction for white and for green.

There is no sign of any mental impairment.

I have seen the patient on and off during the past two years. During this time she has given birth to a third child successfully, and her condition has remained about the same.

We have in this case, to sum up briefly the symptoms presented, an *ataxic, paraplegic gait,* with *cerebellar swaying, increased knee-jerks* and *ankle-clonus;* loss of dexterity and *tremor* in the *hands* upon movement; *slow speech, excessive mimetic contraction* in speaking. In the eyes, *discoloration of the disks,* with amblyopia and somewhat *contracted visual fields.*

The diagnosis of locomotor ataxy is, of course, out of the question. A combined systemic sclerosis (ataxic paraplegia) can be but part of the morbid condition in view of the ocular changes. Friedreich's ataxy can be excluded by the exaggerated knee-jerks, the optic atrophy, and the amblyopia. The case corresponds in every respect to the clinical picture of hereditary cerebellar ataxy depicted by Marie. Despite the lack of direct heredity (a strong neuropathic taint is present), I have always regarded it as a case of this type; but I have never been able to convince myself beyond doubt that it is not, after all, one of those instances of disseminated sclerosis in which the intention tremor is absent or but slightly developed, as in the cases with autopsy seen by Leube, Ebstein, Engesser,[7] Jolly, Gowers, and others. The temporary paraplegia in the legs at the age of nineteen years would rather support this view, although Marie states that remissions also occur in hereditary cerebellar ataxy.

5. In certain cases of *acute disseminated myelitis* and *encephalomyelitis* following the acute infectious diseases the symptoms of an acute or subacute multiple sclerosis are presented, more especially the intention tremor, the increased reflexes, and the scanning speech. The disease may ultimately form the basis of a typical chronic insular sclerosis, with its recurrent attacks, etc. It may, however—and that is a point of importance—subside after a longer or shorter period and end in

recovery. Thus, Leyden[14] mentions the case of a child in which he saw an acute disseminated myelitis develop after an attack of grip, and in which there was perfect recovery; and he cites similar cases in adults reported by Lenhartz and Renviers and himself. Among the cases reported as multiple sclerosis in childhood the only one that could be regarded as belonging to this type is one reported by Massalongo and Silvestri in 1893 in the *Revue Neurologique*, No. 23, in which a child of six years developed intention tremor, scanning speech, increased tendon reflexes, muscular rigidity, spastic gait, and some optic atrophy after an attack of grip. The outcome of the case is not reported. In the presence of a case of this kind it is well to remember that we may be dealing with an old case of multiple sclerosis in which the acute infectious disease has simply rekindled the quiescent chronic disease. Thus, it is not unusual to read in the histories of these cases that the symptoms increased after an attack of scarlet fever, measles, diphtheria, etc. Case I. is a case in kind.

6. *Tumor of the brain.* In an instructive paper in the *Charité Annalen*, 1889, entitled "Ein Irrthum in der Diagnose bei einem 9-jährigen Knaben der das Krankheitsbild einer Multiplen Sclerose bot," Westphal describes a case which he had published a year previously as a case of multiple sclerosis in a child, and in which the subsequent autopsy had revealed a tumor of the left optic thalamus. Gowers states that he has seen typical intention tremor in cases of tubercle in the crus and in the pons, and expresses the suspicion that in some of the cases published as examples of disseminated sclerosis in childhood there may really have been a stationary tubercle of the brain. An error of this kind will be avoided if other symptoms of brain tumor are looked for and found. In our second case the possibility of the presence of a brain tumor was considered; the ptosis some years previous to the development of the other symptoms and the vesical weakness militate against such a diagnosis. Nor was there any history of headache, nausea, or any other general cerebral symptoms. A brain tumor giving rise to a cerebellar ataxic gait would be looked for in the cerebellum or at least in the posterior fossa—a location in which the general symptoms are, as a rule, early and intense.

7. Concerning the exclusion of *hysteria*, it may be briefly stated that the presence of ankle-clonus, of nystagmus (unless congenital), or of optic atrophy excludes it at once. A case of hysteria simulating multiple sclerosis will show indisputable hysterical stigmata if they are only looked for. It may occur, however, that hysteria may complicate the organic disease, which actually happened in one of Bruns' cases, in a girl of fifteen years.

TABLE OF CASES OF MULTIPLE SCLEROSIS IN CHILDREN.

No.	Author.	Sex and age.	Previous infectious disease.	Neuropathic taint.	Remarks.
1	Barthez et Sanné. Traité clinique et pratique des Maladies des Enfants, 3d ed., Paris, 1884.	M. 10	....	......	
2	Idem	F. 6	....	......	Following convulsions from fright. Recovery.
3	Bristowe. Med. Times and Gaz., 1879 .	F. 13	Measles.		
4	Bruns. Berliner klin. Wochen., 1888, No. 5, p. 90.	F. 15			
5	Dreschfeld. Med. Times and Gaz., 1878, vol. i. p. 140.	M. 9	....	Yes.	Brothers.
6	Idem	M. 7	....	Yes.	
7	Drummond. Lancet, 1887, vol. i. p. 12.	M. 8	....	......	Autopsy.
8	Foerster. Jahrb. für Kinderheilk., 1880, Bd. xv. p. 272.	M. 8	....	Yes.	Death; no autopsy.
9	Freund. Arch. f. Psychiat., 1891, Bd. xxii. p. 317.	M. 16	....	Yes.	
10	Ten Cate Hoedemaker. Deut. Arch. f. klin. Med., 1879, Bd. xxiii.	M. 8	....	......	Convulsions.
11	Idem	F. 10	....	......	Fell on head as a baby.
12	Huber. Corresp. Bl. f. Schweiz. Aertze, 1890, p. 43.	M. 7	....	Yes.	Patient's mother had multiple sclerosis; death. autopsy.
13	v. Kezywicki. Deutsch. Med. Wochen., 1892, p. 255.	M. 13			
14	Marie-Charcot. Revue de Méd., 1883, p. 536.	M. 14	... .	......	Recovery.
15	Massalongo and Silvestré. Revue Neurologique, 1893, p. 649.	F. 6	Grippe.		
16	Mensi, E. Riforma medica, 1892, cited in previous article.	?	?	?	
17	Nolda. Arch. f. Psychiat., 1891, Bd. xxiii. S. 565.	F. 9			
18	Oppenheim. Berl. klin. Wochen., 1887, p. 904.	M. 12	Scarlet fever.		
19	Idem	M. 13	Scarlet fever.		
20	Idem . . . . (Neurol. Centralbl., 1890, p. 488.)	F. 14			
21	Pollard. Lancet, 1878, p 183 . .	M. 7	Scarlet fever.	......	Doubtful case.
22	Russel. Lancet, 1889, vol. ii. p. 1120 .	F. 12	....	......	Injury to head.
23	Sachs, B. "Nervous Diseases of Children," Wood, New York, 1895.	F. 14	....	......	Doubtful case.
24	Schwenfeld. Dissertation, Berlin, 1888.	M. 15	Measles, scarlet fever, diphtheria.		
25	Idem	F. 11	Measles, scarlet fever, diphtheria.		
26	Schuele. Deut. Arch. f. klin. Med., Bd. viii. p. 223.	F. 14	Ja'ndice with nephritis at age of six.	......	Death; autopsy.
27	Stadthagen. Jahrb. f Kinderheilk., 1884, p. 1.	M. 11	Diphtheria.		
28	Stieglitz	F. 9	Erysipelas, scarlet fever		
29	Idem	F. 11			
30	Idem	F. 15	Grippe?		
31	Totzke. Dissertation, Berlin, 1893 .	F. 11	Measles, pertussis, pneumonia	Yes.	Sisters.
32	Idem	F. 14	....	Yes.	
33	Unger, L. Ueber Multiple Inselfœrmige Sklerose, etc. Vienna, 1887.	M. 6	....	Yes.	
34	Westphal. Charité Ann., 1888, p. 459 .	M. 11	Pneumonia, diphtheria.		
35	Wilson. Brit. Med. Journ., Nov. 25, 1876.	F. 8	Pertussis.	......	Convulsions; recovery.

ETIOLOGY. Considerable interest attaches to the etiology of multiple sclerosis. Although earlier writers (Westphal, Ebstein, Charcot, Bourneville, etc.) had noted the occurrence of multiple sclerosis in isolated cases as a sequence to typhoid fever, smallpox, cholera, etc., Marie,[19] in 1884, was the first to urge more generally the significance of a previous acute infectious disease in the etiology of the malady. Although received with favor by many writers, Marie's theory has not yet been generally accepted. In 1891 Oppenheim[20] called attention to the frequency with which he elicited in his cases a previous history of metallic poisoning. In a recent publication Strümpell[28] enters a plea for the *endogenic* origin of the disease; he assumes a congenital predisposition in the tissues of the central nervous system, especially in the neuroglia, and regards the various causes assigned by other authors as merely provocative factors which arouse the latent disease.

The etiological conditions obtaining in the cases of multiple sclerosis in children may be summed up briefly as follows: In the first of my cases the first symptoms were referred by the child's mother to an attack of erysipelas in early infancy; after an attack of scarlet fever, at the age of seven years, the child's condition became worse. In Case III. the patient's mother dated the disease from a severe cold, which may have been grippe. In Case II. no etiological data could be made out. In Totzke's series of thirty-five cases collected from literature a history of a previous infectious disease was given in thirteen cases (37 per cent.); in ten cases a neuropathic taint was present. In a somewhat modified series I arrive at about the same figures (14 and 8). As far as we may judge from such small figures it would appear that both these influences are of about equal importance in the etiology of multiple sclerosis.

In conclusion, I wish to add that the prognosis in children is not quite as unfavorable as in adults. Three cases have been recorded by competent observers—Barthez-Sanné, Charcot-Marie, and Wilson—in which the symptoms justified the diagnosis and recovery took place. When we consider the well-known tendency to remissions in the course of the disease, and the fact that certain symptoms may disappear completely and permanently, it is not surprising that this tendency should occasionally result in cure. Of course, a prolonged remission must not be mistaken for a permanent cure.

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MALARIA AS A CAUSATIVE FACTOR IN OTHER DISEASES.

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Spite of all the exhaustive studies on malaria made in this country and abroad, there is still much vagueness in the minds of many practitioners as to what malaria really is and what symptoms or diseases may be justly attributed to it. Malaria is, perhaps, more of a bugbear to the profession at large than any other disease, and were all the evil effects laid at its door truly caused by this disease, it would, next to syphilis, be the disease most to be dreaded in the whole category of ills to which flesh is heir. The ignorant practitioner, unable to find a cause